

# Cyclops Syndrome After Anterior Cruciate Ligament Reconstruction — A Case Report

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## Abstract

Cyclops syndrome is one of the specific causes of loss of extension of the knee following anterior cruciate ligament (ACL) reconstruction. The syndrome is manifested by progressive loss of terminal extension, anterior knee pain and altered gait biomechanics caused by a fibrous nodule of granulation tissue that is located anteriorly along the graft, in the intercondylar notch, starting frequently over 4 to 6 months after the surgery. In symptomatic patients, magnetic resonance imaging (MRI) scan can confirm the diagnosis with high sensitivity and specificity. Management of cyclops syndrome requires arthroscopic excision followed by early and intensive postoperative physiotherapy to regain knee extension. Our purpose is to present a case report of a cyclops syndrome as a complication of an ACL reconstruction performed with an autologous bone-patellar tendon-bone (BPTB) graft in a semi-professional football player, treated successfully with arthroscopic excision and physiotherapy.

**Keywords:** ACL reconstruction complication; Loss of extension; Cyclops syndrome.

## Introduction

Anterior cruciate ligament (ACL) rupture is a common ligament injury of the knee that mainly occurs in pivoting sports such as football, rugby or basketball and could be a devastating lesion for the athlete, causing functional deficits, knee joint instability, increased risk of second ACL injury and long-term joint morbidity. Especially in this group of patients, ACL reconstruction is the treatment of choice to restore knee stability and biomechanics and prevent early degenerative changes of the joint with overall good results. However, as in any other surgical procedure, ACL reconstruction has its own complications. One of the most challenging postoperative complications to treat is limited range of motion of the knee [5].

Cyclops syndrome is one of the specific causes of loss of extension of the knee following ACL reconstruction [4, 5, 6, 7]. The syndrome is manifested by progressive loss of terminal extension, anterior knee pain, altered gait biomechanics and an eventual audible and palpable “clunk” when extending the joint, caused by a fibrous nodule of granulation tissue that can also contain bony or cartilaginous fragments inside [1], called “cyclops lesion”, that is located anteriorly along the graft, in the intercondylar notch. The name “cyclops” derives from its arthroscopic appearance. The fibrous mass appears like a head with a reddish-blue central area of venous channels giving the impression of the eye of a cyclops. The symptoms start frequently over 4 to 6 months after the surgery but cases of delayed onset have also been published. The incidence of cyclops syndrome reported in the literature may vary between 1% and 10% of all the ACL reconstructions [2, 6] and it's the second most common cause of restricted knee extension after graft impingement.

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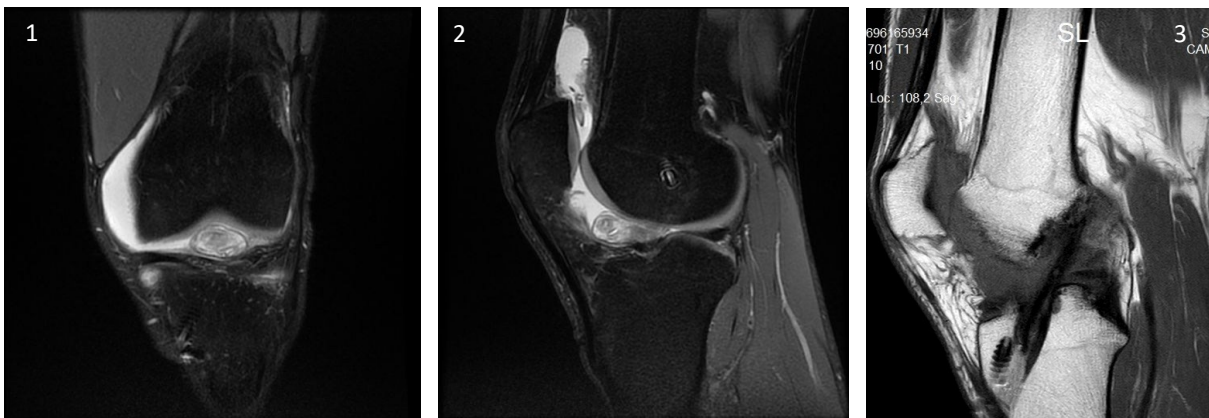
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Some of the risk factors for developing cyclops lesions include high graft volume (BPTB grafts), excessively anterior tibial tunnel placement, narrow intercondylar notch, bony avulsion injuries or bony fragments that remain between the Hoffa's fat pad and the distal attachment of the ACL when drilling the tibial tunnel, and double-bundle ACL reconstruction [2, 4, 6]. In symptomatic patients, MRI scan can confirm the diagnosis with high sensitivity and specificity. Radiographs and CT scans may help in assessing bony fragments and the accuracy of the position of the tunnels. Management of cyclops syndrome requires arthroscopic excision followed by early and intensive postoperative physiotherapy to regain knee extension [4, 5]. We present a case report of a cyclops syndrome as a complication of an ACL reconstruction performed with an autologous BPTB graft in a semi-professional football player, treated successfully with arthroscopic excision and physiotherapy.

## Case Presentation

A 31-year-old semi-professional football player presented with persistent painful loss of extension of his left knee. He had undergone ACL reconstruction with autologous BPTB graft 4 years before, performed by another surgeon. Physical examination demonstrated knee extension deficit of about 10 degrees, anterior knee pain that increased when forcing extension, negative Lachman, pivot shift and anterior drawer test with no subjective knee instability. The patient reported the symptoms started within 6 months following the ACL reconstruction, during the functional recovery phase of the rehabilitation. However, 8 months after the surgery, he returned to play. He could never reach the same pre-injury level, feeling pain and swelling at the end of every game. Anteroposterior and lateral radiographs and a CT scan showed correct placement of the tunnels and a bony fragment in the anterolateral aspect of the intercondylar notch. Magnetic resonance imaging revealed a cyclops lesion with normal signal intensity of the graft. Due to the persistent symptoms and functional limitations, we decided to perform an arthroscopic excision and debridement of the lesion followed by an aggressive physiotherapy protocol that started 1 day after surgery with the aim of regaining full extension and muscle strength. At 15 days postoperatively, we found a complete range of motion, no swelling, good muscle recovery and the patient reported no pain at full extension. He started on-field training sessions 3 weeks after surgery and returned back to competition 45 days after surgery. At 1 year post-op, imaging studies were normal, the patient had his preinjury level of athletics and was asymptomatic.

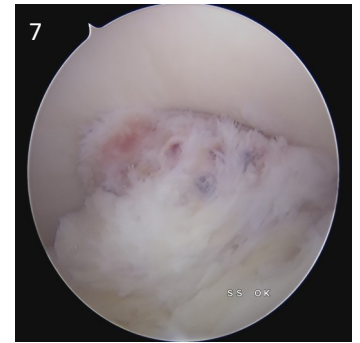
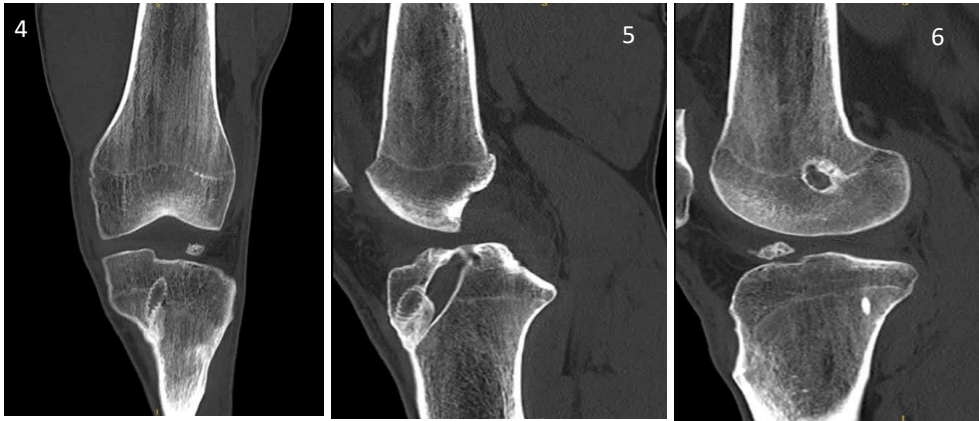


**Fig 1, 2, 3.** Typical MRI findings: Intact ACL graft with Cyclops Lesion.

1: PD FSE FS Coronal View. Cyclops Lesion.

2: PD FSE FS Sagittal View. Cyclops Lesion.

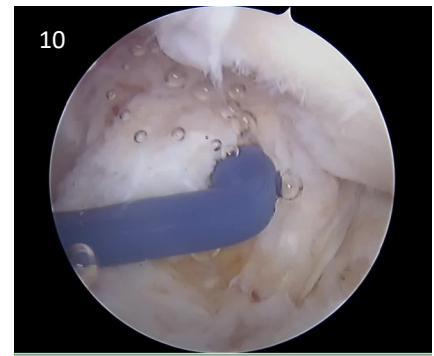
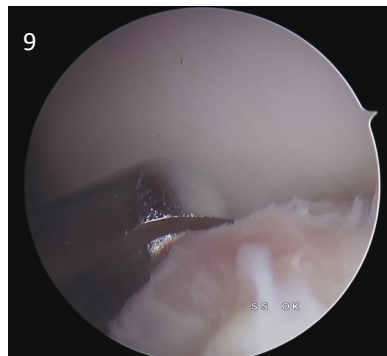
3: T1 Sagittal View. Intact ACL Graft.



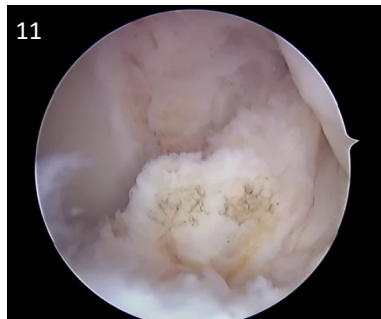
**Fig 7.** Arthroscopic view of the cyclops lesion from the anterolateral portal.

**Fig 4, 5, 6.** CT scan showing correct placement of the tunnels and a bony fragment in the anterolateral aspect of the intercondylar notch.

{- 4: Coronal View. 5, 6:



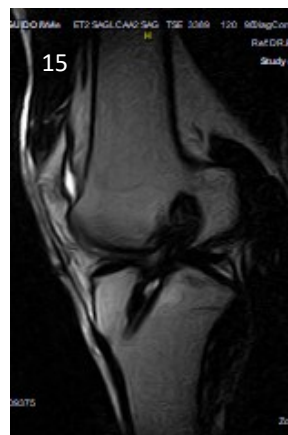
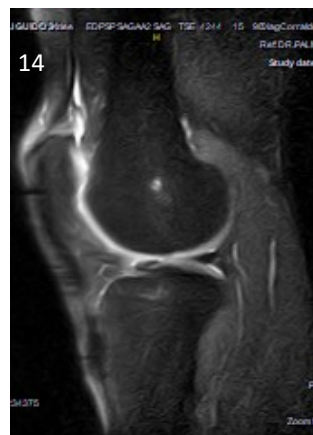
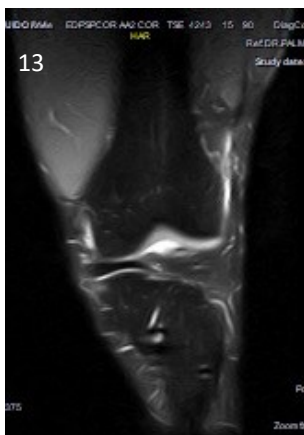
**Fig 8, 9, 10.** Arthroscopic excision and debridement with basket punch, shaver and radiofrequency.



**Fig 11.** Final view of the intercondylar notch and the ACL graft after the excision of the cyclops lesion.



**Fig 12.** Macroscopic view of the excised nodule.



**Fig 13, 14, 15.** Normal MRI at 1 year post-op.

- 13: PD FSE FS Coronal View.

- 14: TSE Sagittal View.

- 15: T2 Sagittal View. Intact ACL Graft.

## Discussion

Cyclops syndrome is characterized by progressive loss of terminal extension and anterior knee pain caused by a fibrous nodule of granulation tissue called “cyclops lesion”, that is located anteriorly along the graft [6], in the intercondylar notch acting as a mechanical extension block of the knee joint. Since Jackson and Schaefer first described the cyclops syndrome in 1990 as a postoperative complication in patients who had undergone anterior cruciate ligament ACL reconstruction with a patellar tendon autograft [1], many articles have been published in the literature about the topic. It's been also reported as a cause of loss of extension after a partial rupture of the anterior cruciate ligament not treated surgically [7], however, this condition is a typical postoperative complication after ACL reconstruction. The exact etiology of the lesion remains uncertain. The origin could be multifactorial and may be related to microtrauma [5, 6] or impingement of residual tibial ACL stump or graft fibers, excessive fibrosis or alternatively due to fibrocartilaginous debris during drilling of the tibia for anterior cruciate ligament reconstruction [4, 5]. No difference has been noted in the location, histologic features, or amount of loss of extension between cyclops lesion after rupture of the native ACL and that after ACL reconstruction [6]. Some of the risk factors for developing cyclops lesions can be classified into pre-operative, such as a narrow intercondylar notch or bony avulsion injuries and intraoperative, such as high graft volume (BPTB grafts), excessively anterior tibial tunnel placement, double-bundle ACL reconstruction or bony fragments that remain between the Hoffa's fat pad and the distal attachment of the ACL when drilling the tibial tunnel. The knowledge of these risk factors may improve the ability to prevent the syndrome [2]. Some of the measures to avoid this complication could be based on minimal invasive surgery, early postoperative mobilization, correct tunnel positioning and drilling, accurate removal of residual debris and delayed surgery, not immediately after the rupture [6]. Persistent painful loss of knee extension following ACL reconstruction even after an accurate physiotherapy program should raise suspicion of a cyclops lesion [3, 6]. In symptomatic patients, MRI scan is the gold standard for evaluating and confirming the syndrome. Other causes of extension deficit such as graft impingement, meniscal tears, hamstring contracture or capsular adhesions and arthrofibrosis must be considered and ruled out. Radiographs and CT scans may help in assessing bony fragments and the accuracy of the position of the tunnels and fixation devices. Once the lesion is confirmed, surgical excision is recommended followed by early and intensive postoperative physiotherapy [3], as they provide good prognosis and have shown to have low recurrence rate [6], preventing long-term biomechanical changes.

## Conclusion

Cyclops syndrome should be always suspected when a patient complains of painful loss of extension of the knee after ACL reconstruction. Magnetic resonance imaging scan is the modality of choice for assessing and confirming this postoperative complication. Symptomatic cyclops lesions can be treated successfully with arthroscopic excision and debridement. An aggressive rehabilitation protocol should start as soon as possible after surgery to regain full range of motion and restore normal knee biomechanics.

## Conflicts of Interest

The authors declare no conflicts of interest.

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